

CHRONIC INTESTINAL STASIS.

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by

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## CHRONIC INTESTINAL STASIS.

The subject of intestinal stasis is a wide one and during the last few years since the important discoveries of Lane, ~~has~~ deservedly attracted ~~much~~ notice, both in medical and surgical practice, as giving rise to symptoms hitherto difficult to explain. The tendency, unfortunately, is to lay all ailments to this cause, and we have to be careful to avoid doing this, without proof.

In this paper I wish to take the subject up from the medical point of view principally. In several of the cases which I shall afterwards quote, the diagnosis of stasis, which was made from the history and from the X-Ray reports, has been confirmed by subsequent operations and by the relief obtained when the cause was removed.

In considering the question of chronic intestinal stasis we must recognise at the onset that there is some confusion in the use of the term, which should really be used to describe a condition of stasis in the intestinal contents. As a matter of fact, the term is more often used to apply to the clinical picture which is considered to result from a state of chronic stasis.

Using the term in the strict sense we understand that there is an unusually long period of time during which the contents of the alimentary tract are retained within the tract.

Lane, using the expression to define the clinical picture, defines the condition of chronic intestinal stasis as one in which there is delay in the passage of the contents of the intestine at one or more points, this delay leading to ~~bacterial~~ infection of the contents with increased decomposition and putrefaction of them, the resulting ~~toxins~~ being absorbed into the circulation in quantities

too great to be neutralized by the ordinary agencies of the body, e.g., the <sup>liver</sup> ~~liver~~ ~~liver~~, and thus causing a general poisoning of the body - "Auto intoxication" - which manifests itself by a degeneration of every tissue in the body and by certain definite clinical characters.

The starting point is thus a delay in the passage of the contents of the alimentary tract.

Gant defines chronic intestinal stasis as a condition characterized by atony or blocking of the intestines, wherein chyme and faeces are intermittently or regularly delayed in their downward passage, so that drainage is interfered with, and bacteria and toxins accumulate to enter the circulation in quantities greater than the emunctories can eliminate, and chronic auto-intoxication ensues.

He also defines constipation as a chronic state of the intestine marked by delayed, infrequent, insufficient or irregular faecal evacuation.

I shall, therefore, in this paper deal with intestinal stasis, and subsequent auto-intoxication. It is one of the most frequent and persistent affections the physician has to deal with, and fully taxes his ability and ingenuity to relieve and cure.

Gant says that the amount of failures in treatment is surprisingly large, and gives as his reasons (1) that physicians too often prescribe remedies or advocate ~~remedies~~ methods which offer temporary relief, because they secure an immediate evacuation, but which accomplish little towards effecting a permanent cure. (2) through ignorance or indifference, they fail to educate the patient as to his manner of living and the proper hygiene of the bowel.

## Anatomy of the intestinal tract.

The small intestine, about 20 feet long, extends from the stomach to the caecum, from which it is separated by the <sup>ileo</sup>caecal valve. It consists of numerous irregularly arranged convolutions, and has a mesentery which is fan-shaped, and thus allows a considerable range of movement. It is divided into 3 parts (1) the duodenum, 10" in length, (2) the jejunum and (3) the ileum. It is larger at the commencement than at its junction with the colon.

The larger intestine extends from the ileo-caecal valve to the anus. It differs from the small intestine in having a nearly constant position, thicker walls, a sacculated contour, appendices <sup>epiploicae</sup> ~~epiploicae~~ and longitudinal bands. The length is between 5 and 6 feet. It is widest at the caecum, diminishing in size towards the rectum, where it again increases considerably, until the anal canal, the narrowest part of the intestine, is reached. It is divided into 4 parts - colon, sigmoid colon, rectum, <sup>and</sup> ~~anus~~.

The colon begins at the caecum in the right iliac <sup>fossa and</sup> ~~psoas~~ at <sup>terminates at the sigmoid colon. in the left iliac fossa, at</sup> the outer border of the left psoas muscle. Beginning at the ileo-caecal valve in a blind pouch (the caecum or caput coli) the colon passes upwards to the liver (the ascending colon) where it describes a sharp turn (the hepatic flexure) and extends across the abdomen to the spleen (transverse colon) where it again turns sharply downward (splenic flexure) as the descending colon, which becomes continuous with the sigmoid colon.

The caecum measures about  $2\frac{1}{2}$ " in both its vertical and transverse diameters, and lies in the right iliac and hypogastric regions at the middle of Poupart's Ligament. It is supported and

held in position by peritoneal folds covering its lateral and anterior walls. It is separated from the small intestine by the ileo-caecal valve, which is designed by Nature and to prevent solids, liquids and gases from entering the small intestine once they have found their way into the large bowel. This valve is a slit-like opening running at a right angle to the ~~only~~<sup>long</sup> axis of the bowel. Its edges are thickened and form lips which are reinforced by strong muscular fibres. As the caecum and large intestines become distended, these valvular folds are firmly pressed together and are so arranged that the contents of the ~~colon~~<sup>ileum</sup> have free exit into the colon, but nothing from the latter can re-enter the small intestine except in rare instances and under great pressure.

Leading off from the caecum <sup>is</sup> ~~to~~ the vermiform appendix, which frequently becomes inflamed as the result of constipation and faecal impaction, and conversely when diseased itself it may cause constipation. It has a mesentery extending about  $\frac{2}{3}$  its length.

The sigmoid colon is about 13" - 17" long, and describes a big S-shaped curve. It is generally considered now to consist of 2 parts (1) the iliac colon extending from the level of the crest of the ilium to the inner border of the left ~~psoas~~ and (2) the pelvic colon starting at the inner border of the left ~~psoas~~ and passing across the pelvis to the right side, thence backward and to the left till it reaches the level of the 3rd sacral ~~vertebra~~, where it forms an acute angle as it joins the rectum. The junction is marked by a distinct ~~constriction~~<sup>increase</sup> in the circular fibres, producing a narrowing of the gut. This muscular ring is known as the Sphincter of O'Beirne. The mesenteric attachment of this part of the colon is short above in the iliac fossa, and below at the junction



with the rectum, but is much larger between these points, and thus this section of the bowel is capable of considerable motion and change of position. It is the narrowest portion of the large intestines.

The rectum is tubular and devoid of muscular bands. The anterior and posterior walls ~~sheu~~ <sup>separate</sup> above the levatores ~~and~~ remain in contact forming a transverse slit, while below this the lateral walls are in ~~ap~~position forming an antero-posterior slit. The upper part of the rectum down to the level of the levatores ~~ani~~ is known as the ampulla. It is capable of being moved laterally or vertically and can be distended by inflation. In the latter case it is seen to be divided into compartments of varying size depending on the number of valves of Houston and the distance between them.

The upper part of the rectum is much less sensitive than the lower part, and surgical interference, ulceration or malignant disease causes comparatively little pain.

The anal canal extends from the level of the ~~the~~ levatores ~~ani~~ and above, to the ~~anus~~ below and is embraced by the internal and external <sup>sphincter</sup> muscles.

The lymphatics of the small intestine are divided into (1) those of the mucous membranes and (2) those of the muscular coat, and they form plexuses. They run between the folds of the mesentery and end in the mesenteric lacteals, and thence pass into the intestinal lymphatic trunk and thoracic duct. They are provided with valves to prevent a backward flow. The lymphatics of the large intestine enter the mesenteric glands, except those of the sigmoid which enter the lumbar glands.

## Physiology of the intestinal tract.

The contents of the small intestines are slowly propelled towards the caecum by a series of peristaltic waves, and in addition another type of movement occurs, viz: segmentation. This is difficult to see in man by the X-Rays, because the passage is so rapid and the large quantity of digestive juices dilute the opaque material. The function of segmentation is to mix the food thoroughly with the digestive juices and to bring every portion in contact with the absorbing mucous membrane. With an opaque meal, in a normal individual, on an average, the caecum is reached between  $3\frac{1}{2}$  and 5 hrs. Up to this time the caecum contains nothing but gas, and perhaps some faecal material adhering to the mucous membrane. The ileum should be completely empty 2-3 hours after the stomach. Observations on patients with caecal fistulae have shown that 350 grammes of fluid material pass through the ileo-caecal valve in one day, whereas the average weight of the faeces is 135 grms. There is therefore a considerable absorption of water between the caecum and the anus, and experiments have also shown that sugar, coagulable protein, and the soluble products of digestion are also absorbed from the colon. Most of this absorption occurs in the caecum and ascending colon, and the contents become less fluid. Further absorption occurs in the transverse colon, where the consistency of the faeces becomes firmer, and they remain the same till they are in the lower part of the sigmoid or pelvic colon, where they attain their normal firm consistency, and are retained until defaecation occurs. The contents of the colon are propelled by a series of peristaltic waves as in the small intestine, but they occur much less frequently and are less efficient on account of the occasional occurrence simultaneously of anti-peristaltic waves. The effect of this is to

cause the contents of the ~~proximal~~ part of the colon to circulate and to come into frequent intimate contact with the mucous membranes so that rapid absorption of water and nutrient material occurs. (All observers do not agree that anti-peristalsis occurs.)

The ~~hepatic~~ flexure is reached from 5 - 8 hrs. after a meal, the splenic flexure between 7 and 14 hours and the junction of the descending with the iliac colon in between 8 and 16 hours. The passage through the pelvic colon is slower, depending upon whether there is a faecal material already there or not. By defaecation the contents of the distal half of the transverse colon enter the descending colon, from which <sup>they are</sup> ~~it is~~ evacuated together with all the ~~faeces~~ already present between the splenic flexure and the anus. Hence if food is taken 9 or 10 hours before defaecation, some of it will be present in the stool. The rest continues to advance till it reaches the pelvic colon and there it remains until the next act of defaecation, which will probably not take place till 24 hours later. Thus if the bowels are opened once a day, the interval between the taking of a meal and the excretion of its residue may be anything from 8 to 32 hours.

The local stimuli which give rise to intestinal movements are mechanical and chemical. It has been shown that mechanical irritation of any part of the intestines causes contraction to occur above and a relaxation below it. This action causes the contents to move onward, so that there is constantly a wave of contraction preceded by a wave of relaxation passing down the intestines. The mechanical stimulation is produced by the direct action of indigestible and undigested remnants of food and by the distension which they produce when mixed with the digestive juices



and the multitude of ~~bacteria~~ present in the colon. A diet containing vegetables with a good deal of indigestible cellulose leaves a larger residue than one consisting mainly of animal food - a point to be remembered in treatment.

Oil increases the movements of the small intestine, and the products of its digestion and ~~bacterial~~ decomposition - glycerine, fatty acids and soaps - are comparatively powerful stimulants, which act on the colon as well as the small intestine.

Exercise stimulates the intestinal movements partly by the increased movement of the diaphragm causing rhythmical variation in the intra-abdominal pressure, and partly by the movements of the abdominal muscles and the ilio-psoas.

The faeces accumulate in the pelvic colon and as the result of mechanical or chemical stimuli, e.g., the taking of breakfast on an empty stomach, active peristalsis of the whole intestine is set up, so that some faeces enter the rectum and this sense of fullness ~~ex~~ gives the desire to defaecate. ~~Afferent~~ nervous impulses pass from the rectum to a centre in the Lumbar cord, and set in action efferent impulses upon which the reflex act of defaecation occurs. If the inclination to defaecate is resisted, the desire soon passes away, and the faeces remain in the rectum until the same hour next day probably. It is this retention of faeces in the rectum that so often leads to ~~diminished~~ sensibility of the rectal reflex, and is one of the commonest causes of constipation. In the colon, toxic substances ~~the~~ developing in the course of abnormal decomposition of proteins are absorbed. These toxins may be due to the influence of prolonged ~~bacterial~~ putrefaction or the presence of abnormal types of bacilli.

Metschnikoff, in his "Prolongation of Life" says that on the one hand animals are shorter lived than birds and lower ~~vertebrates~~ <sup>vertebrates</sup> on the other hand the large intestine is much longer in them than in any other vertebrates, and he wonders if there is here any link of causality, binding the two characters, or is it a mere coincidence. In the lower ~~vertebrates~~ <sup>vertebrates</sup> the large intestine is nothing more than a mere ~~residue~~ <sup>reservoir</sup> for the waste matter in the food, and the caecum ~~alone~~ <sup>above</sup> can be thought to have some digestive property. In many birds the caeca have been found to be rudimentary and in others absent, so that it is to be supposed these organs are useless and are in process of degeneration in this class. In most mammals, e.g., the horse, the caecum is large, and as in most cases the food stays some considerable time in it, it is probable that some process of digestion goes on in it. In many other cases, e.g., the cat and the dog, the caecum is small, in which case its digestive power must be slight.

Metschnikoff asks the question why should the large intestine be so much more developed in mammals than in other vertebrates, and he forms the theory that it has been increased in mammals to make it possible for these animals to run long distances without having to stand still for defaecation. The organ, then, would simply have the function of a reservoir for waste matter.

Although the capacity of the large intestine may preserve an animal in emergencies, such as pursuing its prey or escaping its enemy, it is attended with disadvantages that may shorten the actual duration of life. The accumulation of waste matter, retained in the large intestine for considerable periods, becomes a

aidus for microbes which produce fermentation and putrefaction harmful to the organism. When the faecal matter is free from microbes as is the case with the meconium of the new-born infant, it is not a source of danger to the organism.

The ill-health which follows retention of faecal matter is certainly due to the action of some of the microbes of the gut. It is generally believed that they form poisonous substances which are absorbed by the walls of the intestines and pass into the system. The phrase "auto-intoxication" is based on this interpretation of the morbid processes involved. Attempts have been made to isolate and study the poisons in question and injections of the microbes into the veins of rabbits have produced symptoms similar to those found in obstruction of the bowels and retention of faecal matter.

Some of the products of the intestinal flora are undoubtedly toxic. Butyric acid and the products of albuminous putrefaction are amongst the most pernicious of the microbial poisons produced in the large intestine. Analysis of the urine, in cases of constipation, shows an excess of the sulpho-conjugate ethers, including indican, and which are known to be the products of intestinal putrefaction.

Not only is there auto-intoxication from the microbial poisons absorbed in cases of constipation, but microbes themselves may pass through the walls and enter the blood. About this latter statement there has been much controversy, but it seems highly probable that while the intact intestinal wall offers a substantial obstacle to the passage of bacteria, some do pass through it and enter the blood and the other organs, but whether this is due to

the wall of the gut having been injured or not, it has not been possible to prove.

Metschnikoff therefore infers that the more a digestive tract is charged with microbes, the more it is a source of harm and capable of shortening life. ~~As~~ the large intestine is most richly charged with microbes and is relatively more capacious in mammals than in other ~~vertebrates~~ it is, according to him, a just inference that the duration of life of mammals has been shortened as the result of a chronic intoxication from an abundant intestinal flora. At this stage it will be well <sup>to</sup> consider briefly the organisms and toxins that are found in the alimentary canal normally.

Distaso classifies them as follows :-

1. Non - indol forming organisms.
2. Indol forming organisms.

These are constantly present in the intestinal flora of adults in the normal state: they are also present in the 1st stage of intestinal putrefaction. The flora of the normal adult is certainly harmful, and far from defending the organism against infection, it may be said to help it. In the colon, indol and other bodies belonging to the aromatic series, are elaborated and absorbed, and these bodies when absorbed severely tax the function of the body and therefore of the <sup>liver</sup> ~~liver~~. These bodies, also, are not able to induce peristalsis, in fact they have ~~not~~ an inhibitory action on peristalsis, and may even irritate the mucous membrane. Since these poisons elaborated in the colon, cause auto-intoxication, it would therefore follow, according to this theory, that the exclusion of the colon must be followed by the disappearance

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of constipation and of the resulting intoxication.

He made a series of experiments inculcating (1) normal faeces to which sterile water was added and (2) normal faeces without the addition of water. He then found that in the 2nd case the flora were identical with those in the faeces of constipated persons, i.e.,

- (1) The total number of bacteria was less than normal.
- (2) Gram ~~and~~ negative organisms were almost entirely absent.
- (3) Spores were very abundant.

Bacteria, therefore, in the proximal part of the large intestine are identical with those to be found in the distal part, but in the latter they are far less abundant, and more spores are found (as in experiment (2)). Not only, therefore, is there intoxication by the products of bacterial growth, but the disappearance of bacterial indicates a process of autolysis, the products of which rather when absorbed, cannot fail to be harmful.

If, therefore, the large intestine is excluded, there is an entire change in the intestinal flora, which now resembles the flora present in infants, and it is evident that if these harmful bacteria are removed, the ill-effects of constipation will be removed also - hence, Lane's operation of ileo-sigmoidostomy, as a cure for auto-intoxication. The products of intestinal bacteria, by increasing secretions inhibiting submucous plexuses give rise to stasis. The accumulation of faeces in the colon causes it to fall downward, dragging on the organs which are attached to it. The ileum especially will be affected, and with it the ileo-caecal valve may become inflamed and nearly occluded. Faeces, therefore, will remain longer than normal in the ileum, and it has been noticed by <sup>several</sup> casual observers that in those cases the movements of the large bowel through increased, are irregular and inconstant in direction, in other words



there is hypertony of the colon, and the result is that the faeces are pushed up and down for some time so that they become dried up.

Hence, in constipation, we have on the one hand soluble poisons arising from the intestinal bacteria, and poisons retained by the dead bacteria on the other hand, and these give rise to auto-intoxication.

The toxins in the alimentary canal may now be considered. These may be grouped under different headings:-

1. Foreign bodies ingested in the food.
2. Toxins produced in the tract as a result of decomposition, fermentation and putrefaction, of the ingested food.
3. Bacterial toxins.

Class I. is not within the scope of this paper.

Class III. These have been discussed before.

Class VI. The toxins formed by splitting up of food. In the normal course of food-metabolism various end-products result. Thus from fats we get Lactic, diacetic, oxybutyric acid, acetone etc. These are absorbed and mostly are rapidly altered and combined so as to render them harmless. If they are present in large quantities a certain amount is excreted in the urine. In this case, however, we do not have the symptoms of typical alimentary toxæmia. Proteins are split up into amino-acids and these are absorbed, carried to the <sup>liver</sup> ~~liver~~ and reconstructed. Some of these bodies are known to be toxic and it may be suggested that when absorbed they are responsible for the symptoms of auto-intoxication. However, the mere fact of absorption does not leave the toxic bodies free to produce general effects, because they are carried to the <sup>liver</sup> ~~liver~~, which converts them into harmless bodies. (Should the <sup>liver</sup> ~~liver~~, by disease for example, fail to deal with the toxic bodies, it is possible that auto-intoxication will occur.)

In the bowel the food is <sup>not</sup> ~~also~~ merely subjected to the action of the gastro-intestinal juices, but is also subjected to the action of

the swarms of bacteria present. Many of them, especially the strict anaerobes found in the lower colon are capable of splitting protein bodies to a stage considerably beyond what is the normal end-result of digestion, and it is suggested that these final products of the fermentation, putrefaction and decomposition of proteins by the bacteria of the colon are responsible for toxic symptoms. As a result of experiment, however, it is found that these end-products of bacterial action, viz: skatol, indol, phenol, H<sub>2</sub>S. etc., are not markedly toxic, and moreover, such bodies when absorbed from the bowel have yet to pass through the liver, where they are rapidly rendered harmless.

Herter by the administration of indol, produced in man frontal and occipital headaches, colic, insomnia, lassitude, and after continued administration, a tendency to neurasthenia, but the dose he gave was much greater than the amount which is likely to be absorbed from the intestines, in the severest cases of constipation and the quantity in the urine bears no relation to the severity of these symptoms.

Many cases of neurasthenia present evidence of faulty intestinal conditions and are rapidly relieved of nervous symptoms by the correction of recognised faulty conditions. It is quite probable, therefore, that intestinal disturbances are a factor, if not the universal cause of neurasthenia. A positive organic poison such as we know to result from intestinal putrefaction in a person whose reserves of nervous energy has been depleted, will exert a positive destructive force upon the nerve cell, such as cannot be exerted by any external irritant, e.g., worry or mental distress, and this combined with improper nourishment and fatigue offers quite a reasonable

explanation of nervous breakdown.

Nature has produced a wonderful system of defence against intestinal toxins. Combe says: "The digestive fluids neutralize the digestive toxins; the intestinal epithelium plays an antitoxic rôle; blood returning from the intestines is obliged to pass through the liver, the epithelium of which is endowed with mighty toxicolytic power: antitoxic glands, thyroid, thymus, and suprarenal, modify and neutralize certain toxins of intestinal origin which circulate in the blood; finally the eliminating organs constantly reject and throw out the products of intestinal putrefaction. Ammonia and acetone are eliminated by <sup>the respiration; the skin throws out with the</sup> sweat, indol, phenol and sulpho-ethers; and lastly the kidneys eliminate through the urine the majority of intestinal poisons."

If the system of defence becomes weakened or if the products of intestinal putrefaction become excessive, then toxæmia follows with its symptoms.

The chemical sign of intestinal toxæmia of putrefactive origin that has attracted most attention is the presence of indican in the urine. It is usually present but not necessarily so, and its presence or its amount does not determine the degree of putrefaction or bear a relationship to the amount absorbed. A large amount in the urine or faeces shows that a large amount is being eliminated, but does not show how much is present in the circulating blood. Nevertheless, the presence of indican and other aromatic compounds demonstrate that substances formed during the process of putrefaction have been absorbed.

The amount of disturbances in the body depend upon :-

1. The degree of toxicity of the putrefactive products of the

digestive tract, which have been absorbed and have passed the line of defence unaltered.

2. The length of time through which tissues have been exposed to the action of these products.

3. Tissue resistance or individual cellular reaction.

4. The presence of diseased tissues or organs.

5. The activity of eliminating organs, which will rid the system of these deleterious substances before they produce harmful results.

Effects of intestinal stasis. The intestinal contents in a normal individual are constantly on the move, but if they move more sluggishly or are dammed back by any obstruction, more time and opportunity are given for bacterial action, with the resulting extreme stage of splitting up of proteins into their final end-products. Also, the bacteria have time to spread upwards and so cause a marked increase in the bacterial contents of the lower parts of the small intestine.

It is to this ascending bacterial infection of the lower part of the small intestine as a result of ileal stasis, with the resulting putrefaction of the contents, that Lane ascribed the symptoms and results of auto-intoxication, the toxins according to him being largely absorbed from the decomposing contents of the ileum and not so much the colon. The fact remains to be explained how although in a healthy person faecal stasis cannot produce general toxæmia, with a healthy bowel and healthy organs, yet there may be definite toxic results from chronic constipation.

The explanation probably lies in the condition of the bowel wall itself. Chronic constipation leads to a state of catarrh in the

mucous membrane of the bowel. This simple catarrhal inflammation in very severe cases may even pass on to ulceration or perhaps perforation of the bowel wall.

The bowel wall being thus damaged, it is very probable that toxic substances can pass through in larger quantities than can be dealt with completely by liver, thyroid, etc., and so alimentary toxæmia occur.

As a matter of fact in cases of well marked alimentary toxæmia the bowel wall, especially the colon, is abnormal even to the naked eye.



Granted that the alimentary toxæmia arises from chronic intestinal stasis, the question next arises "What is the cause of stasis?" Many theories have been offered to account for this. On examining the abdomen in these cases one very often meets with various thickenings of the peritoneum forming the mesentery of the bowel, or definite bands, or veil-like membranes, causing kinks and angulations of the bowel. By some these are considered to be inflammatory, but against this are the following facts: (1) The appendix may be normal and yet there may be extensive membranes reaching over the caecum and ascending colon. (2) The vessels run in parallel lines downwards and inwards through the membranes, which one would not expect in the vascularisation occurring in inflammatory adhesions. (3) There is no thickening in the lymphatics running along the course of these vessels, which would most certainly follow inflammation.

Others think they result from Foetal peritonitis, which is improbable.

Lane's theory is that they are evolutionary. While in the erect position all the viscera tend to be displaced downwards, and the normal relation of the structures in the abdomen to each other and to the abdominal wall is upset, in consequence of this several changes take place. The contents of the large intestine being solid and heavy tend to bring down the large intestine first, and this is seen at the termination, where to prevent a tendency to elongation of the pelvic colon and the return of faecal matter into the descending colon, and also to obviate the prolapse of the iliac colon into the true pelvis, definite lines of resistance are laid down. They appear first as streaks on the outer border of the mesentery, and gradually extend along its surface. After a time they

become thicker and more distinct, forming a definite band which later separates more or less completely from the peritoneal outer surface of the mesentery, except at its limits of attachment. The anchoring of this portion of the bowel by this particular collection of acquired or evolutionary adhesions form a kink. Similar bands form on the outer aspect of the mesentery of the descending and iliac colons, becoming more definite at the splenic flexure, where the kink is made more acute by the drag upon it by those acquired adhesions as they contract & pull it upwards. The grip exerted by the acquired mesentery may be so firm as to fix the descending and iliac colon sufficiently to interfere with the free passage of material through this portion of the bowel, so that the intestinal wall is liable to yield in parts forming diverticula.

The same evolutionary developments continue on the outer surface of the caecum, ascending colon and hepatic flexure with the same result. Besides interfering with the free passage of material through the hepatic flexure, these bands may interfere with the lumen of any portion of the ascending or descending colon.

In addition to the bands external to the caecum, and others form on the under surface of it, and help to support it.

The appendix not infrequently gets fixed in these acquired adhesions along the outer or inferior surface of the caecum, and it is made to take its share in supporting the weight of this organ. It may be fixed at the extremity or any point along its length. In the former case there would be interference with the passage of material from it into the caecum. In the latter case, the caecum would be kinked. In this manner material may accumulate in the caecum and appendix, and appendicitis be set up.

In a similar way an acquired mesentery may form between the transverse colon and adjacent loops of ascending and descending colon. These membranes may relieve the stomach of strain for a time, but later they oppose the normal ascent of the transverse colon so that it drops into the pelvis, and interferes with the pelvic viscera. The consequent delay in the transverse colon brings about ileal stasis, duodenal distension, pylorospasm and gastric ulcer.

The caecum is supported in its inner side by the mesentery of the terminal ileum, and when it is loaded, resistances develop on the inner surface of this mesentery over an area of 2" - 3", so that this inner acquired band helps the mesentery to hold up the caecum. This is attached by its broader extremity to the base of the mesentery, and by its narrower extremity to the convexity of the bowel opposite the attachment of its own mesentery to it. In this way it kinks the ileum. This band is best seen in those whose occupation requires them to stand for long periods.

In cases of ileal stasis due to bands or to adherent appendix the small intestine may be dilated and in some cases hypertrophied, and these features are accentuated as the seat of obstruction is approached. The pull exerted by the prolapsed and heavy small intestine puts a strain on the duodenum and hence the kink is frequently found at the duodeno-jejunal junction. Here again resistances are laid down in the form of bands from the outer aspect of the jejunum to the adjacent abdominal wall, and as these contract they no longer allow the normal movement of the bowel with the movement of the body. The obstruction at the duodeno-jejunal junction causes distension of the duodenum, depreciation of its vitality by general auto-intoxication and later the formation of an ulcer with consequent

pylorospasm. There is again accumulation of material in the stomach and resistances are again laid down to prevent the downward displacement of the pylorus, so that adhesions form between it and the under surface of the liver and gall bladder. Again in consequence of strain transmitted through the great omentum by the loaded transverse colon, there is delay in the stomach, chemical changes take place in its contents, and auto-intoxication thus caused affects the mucous membranes and leads to lowered vitality and finally to ulcer.

These represent the mechanical changes resulting from progressive stagnation in the gastro-intestinal tract, but while bands develop in consequence of this delay, a very high degree of stagnation may result from the prolapse of the intestine without formation of bands, and the accumulation in it of material which cannot pass on and which prevents the further passage of faecal material into the over-full bowel. This is especially clear in the case of a huge distended caecum holding back the contents of the ileum, and yet there may be no ileal or appendicular bands.

Such is Lane's theory of a drainage-system completely at fault, the large bowel forming a cess-pool which cannot be properly emptied and infection and stagnation extending up all along the gastro-intestinal tract.

Against his theory are the following points :-

1. In case of tuberculous peritonitis, where the whole bowel is a mass of kinks and angulations no auto-intoxication occurs, and obstruction, even, is not frequent.
2. Many cases showing marked kinks post-mortem have had no auto-intoxication during life.



3. Many of the thin membranes are too fragile to cause obstruction to the lumen of the bowel. It is more probable that the primary fault is in the muscle of the bowel wall, this being incapable of proper peristalsis.

Hertz does not agree with Lane that the most frequent situation of adhesions is round the ascending colon and hepatic flexure, because with the X-Rays he has never found any prolonged delay in the passage of the intestinal contents before the middle of the transverse colon is reached. He also says he has never been able to find at autopsy, or at an emergency operation a loaded caecum, and he therefore does not agree in calling these places a cess-pool.

In cases where there is delay in the bowel, without any appearance of kinking, or organic trouble it is quite possible that the motor activity of the intestine is deficient. This may be due to (1) weakness of the intestinal muscles (2) deficient reflex activity (3) uncontrolled and irregular action.

(1) Weakness of the muscles of the intestine may be congenital, or there may be an inherited tendency to neurasthenia, which is the cause of constipation. The weakness of the muscles may also be caused by neglect of training into proper habits in childhood, or by too frequent use of purgatives, so that the muscles of the bowel become atonic from want of use. It is also found in the aged, the obese (probably due to a fatty infiltration of the muscles) patients suffering from poor condition of the blood, and from fevers and in cancer.

(2) Deficient intestinal activity may also be due to insufficient peripheral reflexes. (a) Because the stimulation is too weak or (b) because the nervous system is in a depressed condition.



Insufficient stimulation occurs (1) when too little food is taken, e.g., in cases of <sup>voluntary</sup> ~~stomach~~ starvation, of starvation due to organic disease high up in the alimentary tract, or starvation due to neurasthenia, or to pain higher up in the tract making the patient afraid to eat. (2) When the food contains insufficient mechanical or chemical exciting agents, e.g., the carefully prepared and cooked food of the civilized peoples of to-day, where little or no indigestible cellulose is left; or the diet of the poorer classes who are obliged to leave out fruit and greens.

In a large class of cases, the passage through the alimentary tract is normal in rate, and no delay occurs till the material reaches the rectum, and there it is held up either owing to (1) there having been a habitual disregard of the call to defaecation which makes the rectum less sensitive and causes it to be distended with faeces and thus to lose its normal tone, or (2) to inefficiency of the voluntary muscles which take part in defaecation, i.e., the muscles of the pelvic floor and the abdominal muscles. In the latter class a frequent accompaniment (or result)<sup>is</sup> ~~is~~ ~~mesenteroptosis~~, which in itself causes stasis and resultant auto-intoxication. In these cases of dysch~~ie~~gia there is generally a history of purgatives having ~~bee~~ lost their effect, and of an action of the bowels only being obtained by means of an enema and these facts are often of help in diagnosis, even without the additional assistance of X-Rays.

I shall now describe Lane's "toxic type" - the victim of auto-intoxication. The patient is more often a woman than a man. She becomes thin, nervous, melancholic, depressed, neurasthenic, and loses all interest in life. Lassitude, muscular weakness and obstinate progressive constipation are present. The skin becomes

thin, inelastic, sallow, wrinkled and pigmented especially those areas exposed to pressure, etc. Pigmentation begins in the eyelids, spreads over the face and neck, axillae, inner aspects of thighs, over vertebral spines, etc., Excessive secretion, often <sup>offensive,</sup> ~~oppressive,~~ occurs, and chronic mastitis, passing into a state of cystic degeneration is also found. The muscles become flabby and lose tone, so that some of the joints, e.g., the knees, especially in the young, are capable of over-extension. The circulation is depressed and the extremities, especially the feet, become cold, livid and often have impaired sensation, almost resembling Raynaud's Disease. The heart muscle is influenced, and in one group of cases the heart is soft, flabby, and the blood-pressure subnormal; while in the other group the left side of the heart is enlarged, the arteries atheromatous, and the blood pressure abnormally high. Anaemia is a frequent accompaniment. Headache is sometimes present, often of great intensity, and even accompanied by vomiting, suggesting brain-tumour.

Sleeplessness, neuritis, neuralgia, rheumatic pains and rheumatoid arthritis are favourite complications. There may be also infection of the mucous membrane & lining of the uterus. Colitis may also be produced as a secondary symptom. The breath is offensive, flatulence and even vomiting may occur, as the digestion gets worse; the gastric juice is hyperacid.

Removal of fat is one of the first symptoms of auto-intoxication and this brings about an appearance of premature senility. There is also a loss of support to the kidneys and the abdominal organs, which become displaced, and by their displacement exaggerate the already existing stasis in the intestinal tract, thereby creating a vicious circle. The loss of pelvic fat causes a backward displace-

ment of the fundus of the uterus, which presses the rectum, and may be one of the causes of dyschezia; or on the other hand the uterus may be bent forward, but the kinking may cause engorgement with subsequent dysmenorrhoea or menorrhagia.

Asthma frequently is proved to result from stasis, and hypertrophy and adenoma of the thyroid, also exophthalmic goitre, are, in Lane's opinion due to the same cause.

## TREATMENT.

Having found by the symptoms and by the X-Rays if possible, that stasis is present, what is the line of treatment to be? Lane advocates operations in practically all cases, the type of operation naturally depending upon the situation of the stasis, because he considers that medical treatment only causes a temporary alleviation of symptoms, and the various so-called "cures" are tedious, dangerous and at the best only palliative. He therefore considers it is the surgeon's duty to facilitate the passage of material through the several portions of the gastro-intestinal tract, thus ~~ab~~viating the mechanical and chemical fault or faults which may develop along its length, consequent on the peculiar mechanical relationship of the individual to surroundings as involved in the complex conditions of the civilisation of the present day. Before an exploratory operation, an examination by X-Rays should be made if possible, as otherwise when the abdomen is opened the bowel is found to be clear (in a patient properly prepared for operation) and it is practically impossible to discover where the stasis is. In some cases an adherent appendix may be damming back the ileal contents, and its removal would leave the ileum freely mobile and able to pass on its contents. On the other hand there are cases where even the removal of an adherent appendix would not relieve the ileal obstruction and shew where an ileo-colostomy alone would solve the problem of how the ileal effluent could be moved on.

The form of obstruction due to an acquired membrane - now commonly called a Lane's kink - ought, in Lane's opinion to be relieved by colectomy, his reasons against a simple division of the

band or bands being as follows :- (1) The membrane is liable to re-form, especially if the patient is obliged to resume the occupation which was largely responsible for its development in the first instance. (2) The free division of those bands and membranes often produces a peritonitis possibly due to the division of ~~lymphatics~~ ~~and~~ which as they are draining a fouled area are likely to possess sources of infection in the material which they carry.

Lane prefers colectomy to ileo-colostomy as giving better permanent results, as there is an earlier convalescence, and less risk to the patient. Especially does he consider it wise in the two extremes viz: a prolapsed large intestine, where no membranes have formed, and ~~an~~ intestine kinked and controlled at many points by lines of resistance.

Lane goes so far as to say that gastro-enterostomy is not advisable, in a case of gastric and duodenal ulcer, even after medical treatment has been tried, unless it can be proved that there is no ileal stasis. He would prefer to do a colectomy in such a case, as in his opinion the relief of the ileal stasis would lead to the healing of the ulcer naturally. The disadvantages of colectomy are:-

1. Frequent irregular evacuations.
2. Regurgitation into the remaining part of the colon.
3. Aperients may still be needed.

In my opinion Lane's treatment is much too drastic. Operation should not be performed until medical treatment hereafter described has been given a fair trial. When, however, there is no doubt about a controlling appendix, or the X-Rays point to adhesions at or near the caecum or ileum, the simple operation of removing the appendix or bands, should be done, followed by medical treatment in 2 or 3 months, depending on the operation, *site & condition of scar*, etc.



This causes less constitutional disturbance to the patient, and in nearly all cases I have found a vast improvement to follow. A patient already showing symptoms of neurasthenia caused by the anto-intoxication from intestinal stasis merely has these symptoms aggravated by the shock of the bigger operation, and even if, surgically speaking, the operation is successful, has 2 or 3 years of misery and ill-health, during which time he becomes despondent, <sup>loses</sup> ~~loses~~ hope, and becomes a sore trial to the patience of the physician.

#### MEDICAL TREATMENT.

When there is delay in the passage through the intestine the resulting constipation is frequently aggravated by neglect of a proper effort ~~an~~ to evacuate the dry faeces, which have collected in the bowel below the splenic flexure. This part of the bowel never being completely emptied, an increasing degree of faecal ~~ob~~struction is produced. Hence, it is most important that an attempt should be made at the same hour every day to open the bowels. Also a call to defaecation later in the day should never be neglected, lest the faeces by remaining in the rectum should impair its sensibility.

#### DIET.

Intestinal stasis can often be completely cured by diet alone, whether ~~of~~ the stasis be due to deficient motor activity of the intestines, or to a dried up condition of the faeces. Even in dyschegia the diet can play an important part in hastening on the contents of the intestine so that they arrive at the rectum in a soft condition and require less force for expulsion.

The chief points about the diet are:-

1. Sufficient food must be taken, e.g., in neurasthenics who imagine they cannot eat. In some stomach cases where food causes pain, it is found sometimes that the constipation is due to the<sup>in</sup>sufficient quantity of food, rather than to any fault on the part of the intestines.

2. Food must be eaten slowly and wellchewed.

3. The diet must contain a good quantity of cellulose, which has an ~~ib~~ititant action on the intestines. It also adds to the bulk of the intestinal contents and produces distension, which in moderation is a stimulus to the intestinal movements.

4. Sugars, organic acids and their salts and the products of carbohydrates must be included.

Vegetables generally fulfil (3) and (4), so should be increased in the diet. Of these the best are greens of any sort, asparagus, onions, parsnips, turnips.

5. The amount of fat should be increased.

6. Wholemeal bread should be taken, because it contains more cellulose than ordinary white or brown bread.

7. Porridge and oatcake, especially when made from the coarser varieties of oatmeal are useful additions.

8. Fruits also contain sugar and organic acid as well as cellulose, hence are good stimulants, with the exception of bananas, which can have no effect. Fruit, preferably uncooked, should be taken at breakfast, lunch and dinner, if possible.

9. Jam and marmalade are of use, on account of the fruit from which they are made, as well as the sugar which they contain.

10. To increase the fat in the diet, butter should be used

plentifully, both by itself and in cooking.

Cod liver oil and olive oil are valuable also and not only keep the stools soft, but are easily digested and very nutritious.

11. Milk puddings have no stimulating action, nor has white bread, or mashed potato.

12. Meat and eggs must be included in the diet, not for their help with regard to the stasis, but to supply the proteins required for the body's daily needs.

13. Plenty of fluid should be drunk - women especially often fail in this respect. A glass of water before breakfast and at bedtime and even before lunch or dinner aids intestinal peristalsis.

14. Sour milk has a stimulating effect in virtue of the lactic acid it contains. Ordinary milk replaces more stimulating diet, and hence has a reputation for being constipating.

15. Weak China tea as a beverage is preferable to Indian or Ceylon tea, as it contains less tannin. It should be taken with milk as the milk combines with the tannin.

#### DRUG TREATMENT.

The dose of a purgative should be so regulated that one daily action of the bowels is obtained, without at the same time robbing the body of fluid or nutritious material, which has not had time to be absorbed. Attempts should be made from time to time to reduce the dose, and it is a good plan to divide the dose into three parts to be taken throughout the day. This causes less griping and violent action of the bowels.

A combination of <sup>some</sup> ~~coals~~ preparation of strychnine and bella-donna with a vegetable aperient is good. The strychnine increases

the tone of the intestine by stimulating Auerbach's plexus, and the belladonna diminishes spasm and regulates the intestinal movements, hence is of value in cases where the bowel is hypertonic, as well as preventing the griping associated with most vegetable aperients.

Vegetable aperients ~~imitate~~ <sup>and</sup> irritate the intestinal mucosa, so give rise to a local reflex in Auerbach's plexus, which results in increased motor activity. Of these the most useful are:- aloes, (which is a slowly acting purgative, but does not lose its effect even after years); cascara sagrada; senna (best used as an infusion of the pods in cold water), castor oil, the most valuable for occasional use, as it does not cause griping, and for muco-membranous colitis.

Saline purgatives are best given in a considerable volume of water, as they do not leave the stomach until they are isotonic with the body fluids. They should be taken on an empty stomach, and should preferably be dissolved in cold water, which is itself a stimulant to intestinal activity.

Saline purgatives produce little or no acceleration of the chyme along the small intestine, the colon being the part of the bowel which they affect most strongly. They are therefore to be recommended when it is desired to produce a complete evacuation of the colon, without interfering with digestion in the small intestine.

Liquid paraffin acts as a lubricant to the intestine and is useful when the faeces are hard and dry, e.g., in dyschezia.

Enemata act by stimulating the movements of the intestines (1) mechanically (2) thermally (3) chemically. Water or normal saline solution or soap and water are the commonest. Glycerine acts as a powerful stimulus, and defaecation quickly follows the injection, though tenesmus is apt to follow. A glycerine suppository

gives a mechanical stimulus.

High enemata may be given under pressure of  $1\frac{1}{2}$  - 2 feet of water, with a long tube and a funnel, and in this way by altering the position of the body, fluid can be injected as far as the caecum. It can generally be retained for several minutes, after which it is expelled, taking with it the contents of the intestine.

Low enemata are given by a Hegginson's syringe, and do not reach beyond the pelvic colon. Rectal enemata are small and do not reach beyond the rectum.

Olive Oil injected by the rectum softens the faeces and makes them less ready to adhere to the intestinal mucous membrane. Hertz however believes that water has as good if not a better effect especially if small quantities are injected at intervals of half an hour. If oil is injected over night, it prevents any absorption of water from the faeces, and soothes the mucous membranes, making the expulsion of the faeces easy in the morning.

With regard to enemata in cases of stasis, a good rule is that the patient should make an effort at the same hour daily. If unsuccessful, he should insert a glycerine suppository and in 20 mins. make an effort again. If this fails to produce an action on 2 successive days, a simple enema should be given on the second day.

Enemata should be used in dyschegia so that the rectum and pelvic colon may be emptied regularly and given a chance to regain their contractile power.

#### Exercise and massage.

Exercise stimulates intestinal movements by producing rapid changes in the intra-abdominal pressure. Hill-climbing, rowing, skipping, swimming and riding bring into activity the diaphragm and



the abdominal muscles, and cause pressure of the thighs on the abdomen.

Special abdominal exercises are useful, but as they have to be done indoors, the stimulating effect of the fresh air on the appetite is lost.

Abdominal massage directly stimulates the intestinal musculature. When an experienced masseur or masseuse is not available, the patient can do it for himself by means of a ball weighing from 3-10 lbs. (This can be made by filling an ordinary tennis-ball with shot, and sewing it up again.)

In cases of visceroptosis, in addition to massage and exercises to strengthen the abdominal muscles, a proper belt is of great value, as it prevents the abdominal muscles being stretched, and gives them a chance to regain their former strength.

In my own experience I have found that when patients have come to me with a history of ill-health, headaches, vague symptoms of "indigestion", constipation, depression, etc. & I have, when possible, had them X-rayed, that there has been stasis at some part of the intestinal tract. If the stasis has been obviously due to a controlling appendix, or bands, I have recommended operation, simply to have this controlling appendix or bands removed. If the incision has been a para-rectal one, which I consider preferable, it is possible to begin massage and abdominal exercises fairly soon - generally in 6 - 8 weeks. The diet is chosen with a view to giving a good deal of residue, and if it is possible I give no medicine at all. Probably at first, a glycerine suppository may have to be used, but in a very short time this can be dispensed with.

On the other hand, where an examination by X-Rays there seems to be no evidence of bands or adhesions, I begin treatment at once. Massage is not available for all, but the ball of shot can be used, and the abdominal exercises can be done by everyone, and if the abdominal muscles are weak, a firm abdominal belt is advisable. As most of my patients are women, I find that a carefully chosen corset is often all that is necessary, but if this is not satisfactory, or is objected to, a Curtis belt is <sup>useful</sup> ~~all~~ that ~~is required~~. With regard to the regular action of the bowels, in such cases I have found the glycerine suppository, followed by, if necessary, a simple enema, all that is required. In obstinate cases, when constipation has been one of the chief symptoms, I have found that medicines are required just at first and I prefer to give a combination of ~~caseara~~, belladonna, and aux vomica, in divided doses during the day, the doses being gradually reduced until the medicine can be dispensed with altogether.

In all cases the diet is much the same as already described, unless colitis is present, or some other contra-indication.

## ILLUSTRATIVE CASES.

In considering 900 cases whose records I have been able to investigate, many of them patients of my own, - cases of faulty metabolism and diseases of the alimentary tract chiefly - I find that 35% have intestinal stasis in one part or other of the tract. This has generally been shown by radiography, and has sometimes been proved at operation, or by the results of treatment applied to remove the stasis. These cases occurred chiefly in members of the better classes whose ~~trase~~ lives are sedentary for the most part, and from whose diet there has been a steady intentional elimination of all non-nutritious particles, so that the bulky residue which is essential to promote action of the bowels is reduced to a minimum. In their diet there is also an undue substitution of protein for carbohydrate material, which results in a decreased formation of organic acids and gas, which are two of the excitive causes of peristalsis. In these latter cases improvement generally followed the increase of cellulose, decrease of meat, increase of fat and a sufficiency of water in the diet. Of these cases 52 - 53% were women, and 47.7% were men. The symptoms had come to a height generally from the 30<sup>th</sup> to the 50<sup>th</sup> year, 26.3% of the cases being from 30 to 40 years of age, and 34% from 40 to 50 years of age.

The chief symptoms complained of in these cases were as follows (many of the patients having numerous and varied symptoms):-

Abdominal pain.. 18%	weariness and tiredness.. 9.9%
general depression.. 3%	headache.. 29%
loss of appetite, nausea and occasionally vomiting.. 41%;	

backache, muscular pains, pains in the joints, arthritis.. 9.6%  
 flatulence and distension.. 26%; constipation.. 83%;  
 mastitis.. 3%; cholecystitis and gallstones.. 2%; neurasthenia.. 34%  
 sciatica, neuralgias and neuritis.. 4%; high blood pressure.. 11.3%;  
 low blood pressure.. 9.9%; colitis.. 9.5%; cardiac irregularity and  
 palpitation.. 2.5%; cirrhosis of liver.. 3%; skin lesions.. 1.2%;  
 anaemia.. 44%; amenorrhoea, dysmenorrhoea.. 6%; loss of weight.. 35%;  
 dropped kidney.. .6%; (only exaggerated cases); mental derangement  
 1.3%; "Indigestion".. .6%; tetany.. .3%; dizziness and tinnitus.. 2.6%;  
 insomnia.. 1.3%; bad taste in mouth.. .3%; heartburn.. .3%;  
 endometritis.. .3%; exophthalmos.. .3%; drowsiness and lack of  
 concentration.. .9%; sore tongue.. .6%; asthma.. .6%.

These symptoms were not confined to patients who had stasis, as naturally they are found in many others, but I have given them as I found they occurred in definite, known cases of stasis.

Chemical examination showed the following conditions in the above mentioned cases:-

indicanuria	8.2%
hyperacidity	29%
hypoacidity	12.8%

X-Ray examination after a barium meal, the bowel having been cleared, 36 hrs. beforehand by the administration of an ounce of castor oil showed stasis or conditions conducive to stasis, e.g. kinks, suggestions of membranes, dropped viscera, etc., as follows:-

1. Stomach. There was delay of more than 5 hours in 32% of the cases. It was dropped in 15%, hypotonic in 12% and dilated in 6%.

An ulcer was present or was suspected in 3.7%.

2. Duodenum. There was evidence of adhesions in 2.5%. It was looped in .1%, showed diverticula in .4% and an ulcer was present or suspected in 4.8%.
3. Ileum. Stasis occurred in 28%. The ileum was adherent or kinked in 10.2%, dilated in .7% and diverticula were seen in .1%. Regurgitation took place in .1%.
4. Caecum. This was adherent or immobile in 17% and dilated in 2%.
5. Appendix. It was adherent in 5% of the cases, was abnormal to the X-Rays in 10%, showed spasm in .7% and stasis occurred in 3.5%. Appendicectomy had previously been performed in 13%, and yet stasis had remained.  
After investigation and in some cases after medical treatment had been given a trial without successful results, appendicectomy was performed in 3.3% cases, or in several of these relief of symptoms followed immediately, though as a rule no real relief was obtained for several months, until dietetic measures had been used, and abdominal massage had been given. The latter could be started at varying intervals after the operation, depending on the particular incision, and the healing of the wound the condition of the abdominal wall, and the tenderness of the abdomen.
6. Colon. Delay in the colon occurred in 60% cases, but in 90% the delay was mainly rectal, and probably due to ~~floating~~ <sup>blunting</sup> or loss of the reflex of defaecation. A loop was found in 1% of the cases, and adhesions were suspected in 3.6%. The colon was dropped in 4%, dilated in .6%



showed diverticula in .3% and was obstructed by bands in .5%. It was hypertonic, or irregular segmentation occurred in 24%.

Gall stones were suspected in .8% cases. The ileo-caecal valve was incompetent in .4%. Ileo-colostomy had been performed in 1% cases, and the symptoms or X-Rays appearances of stasis still persisted. Colostomy had been done in .5% without relief of the symptoms.

A common history was of constipation, flatulence, headache, etc., extending over several years, and gradually increasing ill-health. This ill-health became as it were acute after a year or two of war-time conditions and was then ascribed by the patient to nerve-strain, war-bread, etc. The tendency to neurasthenia certainly became more marked. After it had been found by X-Rays that stasis was present, either owing to ~~visceroptosis~~ along of the bowel, or the presence of adhesions, or bands, treatment was begun. Dietetic measures were used, as before described. Regular rests and exercise were prescribed and abdominal massage was given, either dry, or under hot water, with special ~~abdominal exercises~~ attention to the parts affected. In many cases abdominal exercises were done. Intestinal douches were given with success, daily or three times a week at first, the frequency being reduced gradually. The faradic current to the colon was also given in several cases, but was not found to be so successful as other methods. Drugs were avoided as far as possible, the patient being recommended to make a daily effort if possible, or if, even with a glycerine suppository no movement of the bowels occurred, an enema was given every other day.

Where these measures failed, a simple cascara pill or saline powder was used, and in time the patient could generally dispense with these also. In some of the very obstinate cases, Liquid Paraffin was found useful.

Where, however, adhesions or an abnormal appendix seemed to be definitely causing the symptoms, laparotomy was performed, and the bands freed, and the appendix removed. In no case was the colon resected, or was ~~it~~ a short circuit done, and improvement generally followed the simpler operation, after an interval of some months.

#### A few typical cases of stasis:

Mrs. B., a woman of 52 years had had flatulence, constipation, and frequently recurring attacks of colitis for 18 years. She had rheumatoid arthritis, incipient cataract, and took frequent attacks of urticaria and angioneurotic edema. Some years previously she had had hysterectomy performed for fibroid, and double oophorectomy. There was now a hernia through the scar of this operation. With the X-Rays she was found to have ileal stasis of 8 hours, the appendix filled normally, and there was delay in the colon of 96 hours, accompanied by hypertony. A diet suitable for constipation was given, containing no milk and abundance of fruit and greens, and massage was given to the abdomen. For a time she improved, and then had a bad attack of colitis. The diet was changed to one suitable for colitis and intestinal douching was begun. The condition did not improve, however, and the patient would not consider operation.

Under the circumstances I believe that even relief of the hernia would have helped the condition, and would have allowed massage to be administered more efficiently.

C., a lady of 65 years had complained of constipation for 25 years, and had been in the habit of taking aperients daily. Her chief complaint was of tiredness and a realisation of her age in spite of very careful living. She was an extremely well preserved woman and one who led a useful but not over strenuous life. X-Rays showed ileal stasis of  $5\frac{1}{2}$  hours, but nothing suggestive of adhesions or organic lesion. She was given massage to the abdomen, both ~~day~~ and under hot water. During 6-8 weeks of treatment she made wonderful improvement, took no aperients but had a daily action of the bowels. She has since resumed her former activities, feels years younger and no longer complains of lassitude.

F., a man aged 37, had complained of indigestion, with flatulence, pain, nausea and constipation for 8 years. He had been in the Army and the symptoms were now getting worse. On examination the gastric juice was found to be hyperacid. With the X-Rays the stomach was seen to empty in 4 hours, but chyme was still in the ileum 24 hours later. The appendix was dilated, and immobile. Appendicectomy was advised and at the operation the X-Ray findings were confirmed, the appendix being reported to be bound down by adhesions. The patient made a good recovery and when heard of a few months later was free from the old symptoms.

M., a woman, had complained of constipation for years accompanied by abdominal pain and distension, and now at the age of 51, suffered from

tiredness, loss of weight, increase of abdominal pain and distension and piles. The gastric juice was normal. Examination by X-rays showed the stomach taking 6 hours to empty. The ileum was empty 4 hours after the stomach (which is very slightly above normal). The caecum was mobile, and the appendix patent, but tender at the tip. The colon had not emptied in 72 hours, and an enema was then given on account of the discomfort. While under treatment, there was a definite mild attack of appendicitis. Operation was not considered, as patient was highly nervous, and there was no immediate urgency. Dry massage was given to the abdomen, the region of the appendix being avoided. A dry diet was taken containing little milk, but abundance of fruit, and greens. A suppository was used daily, and if it failed a small water enema was given. On this treatment there was great improvement, and at the end of 6 weeks an action was obtained nearly every day, much to the patient's astonishment. The general health was also much better and has remained so, although there have been on several occasions slight relapses to the former constipation.

M., a man of 58 years had suffered from flatulence and constipation since his youth. Duodenal ulcer had been suspected several times, but careful examination had failed to confirm the suspicion. He also suffered from rheumatism and was markedly neurasthenic. The stomach contents were hyperacid. The stomach was found by X-rays long and to be/dropped, but emptied in 5 hours. The colon was markedly prolapsed, took 72 hours to empty and was hypertonic. He was kept in bed for 2-3 weeks, with the foot of the bed raised. A diet suitable for hyperacidity containing less carbohydrate than usual was

taken. Massage was applied daily to the abdomen. On this treatment he improved, and now feels better than he has done for years.

R., a man of 30, had complained of flatulence, distension and constipation for 5 years. The gastric juice was found to be hyperacid, and the stomach took 5 hours to empty. The ileum still contained chyme four hours after the stomach was empty. The lumen of the appendix was irregular in outline, the appendix was fixed at the tip, and still contained barium 60 hours after the commencement of the meal. Appendicectomy was advised, and an appendix found and removed, confirming the above findings. The man went to India 4 months later and had no return of symptoms when last heard of.

S., a woman of 24, had attacks of sickness at varying intervals for years. These were now becoming more frequent, following a period of great mental strain. She had always been constipated and noticed the sickness tended to be associated with failure of a daily action of the bowels. Sleeplessness was also a troublesome symptom. The gastric contents were found to be hyperacid and the urine contained indican. With the X-rays the appendix appeared fixed at the base, and there was 104 hours delay in the colon. The abdomen was opened and the appendix appeared normal in position and mobile. It was removed, however, and on being slit open, several small submucous haemorrhages were found. Microscopical examination showed fibrotic changes. Two months after the operation massage to the abdomen was begun, associated with abdominal exercises. The stomach contents being still hyperacid, a diet suitable for hyperacidity was taken. After several weeks of this treatment there was distinct improvement, although the sleeplessness was no better. The



bowels tended to move more regularly, and there was only one attack of sickness, which was directly traceable to an indiscretion of diet.

#### SUMMARY & CONCLUSIONS.

1. Chronic intestinal stasis is a common cause of ill-health. It is characterised by atony or blocking of intestines, wherein *chyme* and faeces are intermittently or regularly delayed on their downward passage, so that ~~drainage~~ is interfered with <sup>and</sup> ~~or~~ bacteria and toxins accumulate to enter the circulation. In order to excrete these toxins the liver and kidneys are severely taxed and in time they also break down. The toxins exert a deleterious effect on the nervous system, which is rendered unable to sustain any strain and a state of neurasthenia ensues - hence many cases of chronic intestinal stasis have become markedly neurasthenic during the way.

2. According to Lane, the tendency to stasis arises from the formation of bands which develop between the abdominal organs. These he regards as developing during man's changing to the erect position in the course of evolution. They are not found in all cases, but in his opinion are further aggravated by various occupations, e.g., an occupation which requires much standing. These bands are generally found to begin near the lower part of the tract, and by dragging on the intestine, or by forming kinks, cause stasis and a damming back of intestinal contents from which the absorption of toxins takes place.

3. Chronic intestinal stasis is said by Lane to be commoner in women than in men. In my series of cases I find that it occurred

almost as frequently in men as in women,

(In collecting the above facts I have confined myself to the chronic cases. Those which show attacks of acute or subacute obstruction I have purposely omitted, as I consider they can generally be placed in a different category and the symptoms are typical of obstruction.)

2. Patients suffering from headache of whose origin is not obvious, lassitude, difficulty of concentration, insomnia, neurasthenia, flatulence, abdominal pain, constipation, colitis and indigestion, should be examined systematically and whenever possible X-ray photographs should be taken after a barium and buttermilk meal, to see if there is stasis, and if this is present they should be treated according to the cause and the situation of the stasis.

5. Treatment may be (1) medical or (2) surgical. The former consists in regulating the diet, the formation of good habits, exercise in the open air. Massage, and a supporting belt may also be tried. If necessary a suppository and enema may be used, and medicines should be administered only after these remedies fail. Of the medicines, the best are paraffin, cascara with belladonna and strychnine (unless there is a hypertonic bowel) and senna pods.

Surgical treatment should consist of removal of an abnormal appendix and the separating of all membranes and bands. Some Lane advises the removal of the whole colon, as he considers the caecum a cess-pool from which absorption takes place more rapidly even than further down the tract, where absorption is slower, the intestinal contents being less fluid.

6. While surgical treatment is often the only really satisfactory hope of relief, I consider that It should not be undertaken too lightly, as in my opinion the shock to the whole system is much more severe than is generally believed. While, of course, medical treatment with a controlling appendix or a definite kink caused by a membrane, is merely wasting time, I consider that these, when diagnosed, should be relieved before commencing medical treatment. On the other hand, my experience of the after results of colectomy and even of ileo-colostomy is small, but of those cases I have seen the majority have been unsatisfactory, either on account of intractable diarrhoea in the former class, or on account of a caldesac forming in the right iliac fossa, leading to ~~severe~~ toxaemia, with ~~ag~~anosis and great weakness and depression. Therefore, I consider that all other means should be given a long and thorough trial, before anything so drastic is done. (Lane would rather do a colectomy than a gastro-enterostomy in a case of gastric or duodenal ulceration, where he knew there was definite stasis. In theory this would appear to be correct, but in my opinion is too drastic.)

As in the case of all abdominal operations, I consider the patient should be well warned to consider himself or herself a semi-invalid for 9 to 24 months (according to the operation done).

There would then be less of the disappointment with subsequent depression, aggravating the already poor state of health.

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